COMPARISON OF EFFICACY OF ONDANSETRON AND GRANISETRON IN PREVENTION OF POST – OPERATIVE NAUSEA AND VOMITING AFTER TONSILLECTOMY AND MIDDLE EAR SURGERY

Dissertation submitted in partial fulfillment of the requirements for the degree of

M.D. (Anaesthesiology)
Branch X

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CERTIFICATE

This is to certify that **Dr.V.GNANA GANESH**, has prepared this dissertation titled "**COMPARISON OF EFFICACY OF ONDANSETRON AND GRANISETRON IN PREVENTION OF POST – OPERATIVE NAUSEA AND VOMITING AFTER TONSILLECTOMY AND MIDDLE EAR SURGERY**" under my overall supervision and guidance in Madras Medical College, Chennai in Partial fulfillment of the regulations of The Tamilnadu Dr.M.G.R. Medical University, for the award of M.D. degree in Anaesthesiology.

Dr.Kalavathy Ponniraivan M.D.,
The Dean
Madras Medical College,
Chennai – 600 003

Place : Chennai.

Date :

CERTIFICATE

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Dr.Sivarajan, M.D.D.A.,
Professor and H.O.D
Department of Anaesthesiology
Madras Medical College
Chennai – 600 003.

Place : Chennai.

Date :

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INTRODUCTION

Postoperative nausea and vomiting are multifactorial in etiology. It remains a common problem after general anesthesia and contributes to patient dissatisfaction. In a study conducted by Eberhart et al, nearly 50% of patients mentioned PONV as the postoperative side effect of greatest concern[1].

But, much importance has been given to post-op pain relief than to prevention of post-operative nausea and vomiting. When severe, post-op nausea and vomiting can lead to wound dehiscence, bleeding, dehydration, electrolyte imbalance, delayed discharge from PACU, and increased treatment cost to patients.

AIM OF THE STUDY

The aim of this study is to compare the effect of ondansetron and granisetron in the prevention of post-operative nansea and vomiting in patients undergoing tonsillectomy and middle ear surgery and to evaluate the safety of the drugs by studying the incidence of side-effects.

PHYSIOLOGY OF NAUSEA AND VOMITING

The syndrome of nausea, retching and vomiting is known as sickness and each of it can be distinguished as a separate entity.

Nausea : It is a subjective sensation of the desire to vomit, but without any attempt at expulsive movements.

It is frequently accompanied autonomic phenomenon, resulting in objective signs such as secretion of saliva, sweating, increase in pulse rate, variations in rate, depth and regularity of respiration, pallor, and pupillary dilation.

Retching and vomiting: Both are active exclusive mechanisms, and are differentiated by the end result of the process. Vomiting results in forceful expulsion of gastric contents through the mouth, whereas retching causes no expulsion.

Mediators of vomiting reflex :

Vomiting is initiated by an afferent pathway which carries signals from both the viscera and certain areas of brain. These impulses are transmitted to specialized centres in the

brain. Appropriate motor reactions then occur to cause the vomiting act.

Afferent

1) Visceral receptors
2) CTZ
3) Vestibular system
4) Cortex
5) Glossopharyngeal stimulation

Central integrative mechanism

1) Vomiting centre
2) Nucleus tractus solitarius

Efferents

1) Cranial nerves to upper GIT
2) Spinal nerves to diaphragm and abdominal muscles

Afferent pathway

1. Visceral receptors :

Chemoreceptors They are located in upper G1, and respond to GI irritation which release 5–HT from entero chromaffin cells.

Mechanoreceptors :

Found in the muscle layer of GI. These receptors respond to distention, contraction of GIT and physical damage by surgical stimuli etc.,

Both these receptors transmit signals through the vagus and sympathetic afferents via the spinal cords.

2) CTZ :

The chemoreceptor trigger zone (CTZ) is located in the area postrema in the floor of the fourth ventricle. It responds to blood borne drugs, like opioids, digoxin, mediators.like dopamine, 5-HT, histamine harmones, toxins etc., because it is unprotected by the blood – brain barrier.

3) Vestibular system :

When body is rotated or equilibrium is disturbed or when ototoxic drugs act, the vestibular apparatus sends signal to the vomiting centre via the cerebellum.

4) Cortex :

Various psychic stimuli like bad odour, ghastly sight, severe pain, fear, recall of an obnoxious event cause nausea and vomiting through higher centres.

5)Glossopharyngeal stimulation :

Stimulating the back of the throat stimulates vomiting centre via the nucleus tractus solitarius.

Central intergrative mechanism :

Vomiting occurs due to stimulation of vomiting centre situated in the medulla by the afferent impulses reaching it via the nucleus tractus solitarius. Head injury raised ICT, psychic stimuli and vestibular impulses directly stimulate vomiting centre.

Efferent Pathway

The motor impulses that cause actual vomiting are transmitted from vomiting centre through the 5^{th}, 7^{th}, 9^{th}, 10^{th}, and 12^{th}, cranial nerves to the upper GI and through the spinal nerves to the diaphragm and abdominal muscles

1) Antiperistalsis, the preclude to vomiting :

Antiperistalsis may begin as far down in the GI as the ileum, many minutes before vomiting appears. The waves travel up the intestine at a rate of 2 – 3 cm/sec, pushing the intestinal contents to the stomach and duodenum within 3 – 5 minutes. Just prior to vomiting, strong contractions occur in the stomach and duodenum along with partial relaxation of LES, allowing the vomitus to begin moving into the oesophagus.

2) The vomiting act :

Once the vomiting centre has been sufficiently stimulated and the vomiting act instituited the effects are 1) a deep breath, 2) raising the hyoid bone and larynx to pull the upper oesophageal sphincter open 3) closure of glottis 4) lifiting of soft palate to close the posterior nares, 5) downward contraction of diaphragm and simultaneous contraction of abdominal muscles, compressing the stomach in between and 6) finally the LES relaxes completely, allowing expulsion of gastric contents through the mouth.

RISK FACTORS FOR PONV[2-5]

1) Patients related

Age : incidence is low in infants, gradually increases towards adulthood and then decreases again in the elderly.

Sex : Incidence in high in women, especially during ovulatory and luteal phase[6-7].

Anxiety : Causes decreased gastric emptying. Anxious infants swallow air during induction and vomit due to gastric distention. Pain and stress release catecholamines which stimulate area postrema.

2) Anaesthesia related :

Atropine : Decreased gastric emptying.

Opioids : Directly stimulate CTZ through opioid receptors. However, if opioids are not given, it still causes vomiting due to increased pain.

Intravenous anaesthetics: Ketamine, etomidate based anaesthesia have been proposed to cause more PONV than thiopentone and methohexitone. Propofol and midazolam have been shown to be associated with less PONV[8-11].

N_2O: May cause increased PONV due to increase in middle ear pressure, bowel distention and sympathetic stimulation[12, 13].

Volatiles : Due to sympathetic stimulation ether, chloroform and cyclopropane are more frequently associated with PONV than fluorinated anaesthetics.

Neostigmine : may increase PONV due to increased gut motility[14,15].

Neuraxial blockade : Cephalad migration of opioids stimulate area postrema[16]. High blockade, due to unopposed vagal tone, increase GI peristalsis and cause vomiting[17]. Atropine is effective in this situation[18,19].

PHARMACOLOGY OF ANTI – EMETIC DRUGS

Classification

1. Anticholinergics : Hyosine, Dicylomine

2. Antihistaminics (H1) : Promethazine, Diphenhydramine, Dimenhydrinate, Cyclizine etc.,

3. Neuroleptics : Chlorpromazine, Haloperidol, droperidol.

4. Prokinetic : Metaclopromide, Domperidone, Lisapride, mosopride

5. $5HT_3$ antagonists ondansetron, granisetron, dolansetron, etc.,

6. Adjuvants : Dexamethazone, Benzodiazepines, Cannanbinoids

7. Non - Pharmacologic : Acupuncture

Anticholinergics :

Hyoscine (0.2 – 0.4mg oral, i.m) and dicyclomine (10 – 20mg oral) are useful for the prophylaxis of motion sickness. They probably act by blocking conduction of nerve impulses across a cholinergic link in the pathway leading from vestibular apparatus to the vomiting centre. However hyoscine produces sedation and other anticholinergic side effects. A transdermal patch containing 1.5mg of hyoscine, delivered over 3 days, produce only mild side effects[20].

H_1 Antihistaminics :

They are useful mainly in prophylaxis of motion sickness. They are also used in the prevention of post op emesis[21, 22]. The effect appear to be based on anticholinergic, antihistaminic and sedative properties. By their anticholinergic action, they block the extrapyramidal side effect of metaclopramide, while supplementing its antiemetic action. It is better to avoid them for morning sickness, as they have been suspected to have teratogenic potential.

Neuroleptics :

These are potent anti-emetics, act by blocking D2 receptors in CTZ and are effective in PONV, radiation and chemotherapy induced vomiting, and morning sickness. They are not effective in motion sickness, the vestibular pathway does not involve dopaminergic link. Main side effects are sedation and acute muscle dystonia, especially in children.

Droperidol:

It is a butyrophenone neuroleptic, used for the treatment and prevention of PONV. Larger doses of droperidol (>20μg/kg) results in dyskinesia, restlessness and dysphoric reactions. So the lowest effective dose should be given after induction of anesthesia for anti-emetic prophylaxis[24]

Prokinetic drugs

1) Metaclopramide :

It is chemically related to procainamide and acts through both dopaminergic and serotonergic pathways.

a) D2 antagonism

Metaclopramide blocks D2 receptors and increase Ach release in GIT, thereby promoting gastric emptying and LES tone. The central D2 blocking action on CTZ is also

responsible for its anti-emetic activity. However, it has no chlorpromazine like neuroleptic action, though it shares the extra – pyramidal and hyperprolactineamic actions of CPZ.

b) 5 – HT4 agonism :

Activation of 5 – HT4 receptors on interneurones, promote Ach release from primary motor neurons innervating the smooth muscles. This promotes gastric hurrying and improves LES tone.

c) 5- HT3 antagonism :

At high concentrations, it blocks 5 – HT3 receptors present on inhibitory myenteric interneurones, which increase GI emptying through release of non – adrenergic non – cholinergic (NANC) neurotransmitter.

Pharmacokinetics and Interactions

It is rapidly absorbed orally, enters brain, crosses placenta and secreted in milk. It is partly conjucated in liver and excreted in urine within 24 hours. t½ is 3- 6 hours.

It hastens absorption of aspirin, diazepam by facilitating gastric emptying and reduces absorption of digoxin, cimetidine. By blocking DA receptors in basal ganglia, it abolishes the therapeutic effect of levodopa.

Side effects :

Sedation, dizziness, diarrhoea, muscle dystonias in children. Long term use causes parkinsonism and galactorrhoea.

Dose : 0.3 – 1.0 mg/kg im or iv

10 mg tds oral

The combination of metaclopramide 10-20mg iv and droperidol 0.5-1mg iv appears to be more effective than droperidol alone[25,26].

2) Domperidone :

It is a D2 antagonist, chemically related to haloperidol.

Mechanism of action :

It is based on D2 receptor blockade in upper GI, promoting gastric emptying. Unlike metaclopramide, its prokinetic actions are not blocked by atropine.

It crosses BBB poorly, extrapyramidal side effects are rare. It does not abolish the therapeutic effect of levodopa. However, it acts on CTZ, which is outside BBB.

Pharmacokinetics :

Well absorbed orally, but bioavailability is only 15% due to first pass metabolism. It is completely biotransformed and metabolites are excreted in urine. t½ is 7 - 8 hours

Side effects :

Dry mouth, loose stools, headache, rashes, galactorrhoea and rarely cardiac arrythmias on rapid iv injection.

Dose : 10 – 40mg (Children 0.3 – 0.6mg/kg) tds.

3) Cisapride and Mosapride

Both are mainly prokinetic drugs, promoting gastic emptying and LES tone, with little anti-emetic property as they have no action on D2 receptors.

Mechanism of action

Prokinetic action is mainly exerted by 5 – HT4 agonism and 5- HT3 antagonism promoting Ach release from myentric neurons.

Both lack D2 antagonism, devoid of action on CTZ, does not produce extrapyramidal symptoms or hyperprolactinemia.

Pharmacokinetics:

Oral bioavailability is 38%. Inactivated by CYP 3A4 with a t ½ of 10 hours. Dose should be reduced in liver disease.

Side effects:

Abdominal cramps. Diarrhea, dizziness, rise in serum transaminases. At high concentrations, cisapride by blocking delayed rectifying k^+ channels in heart, prolongs Q-TC interval and predisposes to Torsades de pointers and VF. This is not seen with mosapride.

Dose:

Cisapride: 10-20mg tds. Orally.

Mosapride: 5mg tds. Orally.

Adjuvant antiemetics

1). Corticosteroids:

Dexamethasone 8-20 mg iv can reduce nausea and vomiting due to chemotherapy and augment the efficacy of other primary anti-emetics like metaclopramide and ondensetron[27].

2). **Benzodiazepines:**

They have weak anti-emetic property, based mainly on sedative action[28,29]. Used as adjuvant to other anti-emetics, they reduce anxiety and produce amnesia for the unpleasant procedure. They also suppress dystonic effects of metaclopramide.

3). **Cannabinoids:**

Tetrahydrocannabinol (^{9}THC) is the active principle of the hallucinogen cannabis indica. The disorienting and other central effects limit its clinical utility. Dronabinol is less hallucinogenic and more anti-emetic than ^{9}THC.

4). **Neurokinin-1 antagonists:**

They are effective in both the treatment and prevention of PONV. These compounds may act synergistically with $5HT_3$ antagonists.

Non-pharmacologic methods:

There is considerable interest in acupuncture for its potential to prevent PONV with minimal side effects and expense. However, they have limited effectiveness. Available

evidence suggests that acupuncture prevents PONV compared with placebo.

Most studies use P_6 (Nei-guan or pericardium) point[31], located between palmaris longus and flexor carpi radialis tendons, 4cm proximal to distal wrist crease and 1cm below the skin. It is recommended that stimulation be initiated before induction. Some report taping a small needle cap over the P_6 point as a means of acupressure stimulation[32-34].

More recently Korean hard acupressure was shown to be effective in PONV in adults and children.

PHARMACOLOGY OF $5HT_3$ ANTAGONISTS

Ondansetron:

It is a carbazole derivative, introduced in the early 1990's. It is the prototype of new class of anti-emetic drugs developed to control chemotherapy and radiotherapy induced vomiting and PONV.

Mechanism of action:

A.Cytotoxic drugs and radiotherapy produce nausea and vomiting by causing cellular damage → release of mediators including 5HT from intestinal mucosa → activation of vagal afferents in gut → impulses to NTS and CTZ. Ondansetron blocks emetogenic impulses both at their peripheral origin and central relay. It does not block dopamine receptors.

b. A weak gastrokinetic action due to $5HT_3$ blockole.

c. A minor $5HT_4$ antagonistic action.

Pharmacokinetics:

Oral bioavailability is 60-70% due to first pass mctabolism. It is hydroxylated by CYP 1A2, 2D6 and 3A, eliminated in urine and faeces, t1/2 3-5 hours, duration of

action 4-12 hours. No significant drug interactions have been noted.

Dose and efficacy:

1. For chemotherapy induced vomiting: 8mgiv by slow injection over 15 minutes ½ hour before chemotherapy, followed by 2 similar doses 4 hours apart.
2. For PONV: 4-8mg iv (0.1-0.15mg/kg) during induction and repeated if needed after 8-12 hours[35-42].

Patients who do not obtain optimum protection, benefit from addition of dexamethasone, promethazine or diazepam. Adjuvants are more often required for delayed phase vomiting that occurs on second to fourth day.

Availability :

4-8mg tablets

2mg/ml injection in 2ml, 4ml ampoules.

Side effects:

Generally well tolerated. Only common side effect is headache. Mild constipation, diarrhea, abdominal discomfort, rashes and allergy (after iv injection) can occur.

Granisetron:

It is an indazole derivative, 10-15 times more potent than ondansetron. The mechanism of action is similar to ondansetron, except that the weak $5HT_4$ blockade has not been detected in granisetron. t1/2 is 6-8 hours. Side effect profile is similar to ondansetron. It is useful in chemotherapy induced vomiting and post op nausea and vomiting[43-46].

Dose:

20-40 µg/kg.

Availability:

1mg, 2mg tablets.

1mg/ml injection (1,3ml ampoules)

REVIEW OF LITERATURE

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MATERIALS AND METHODS

This is a randomized double blinded study conducted at Madras Medical College. A total of 60 patients posted for tonsillectomy and middle ear surgery under general anesthesia were included.

Inclusion criteria: ASA I and II

No history of motion sickness or prior PONV.

No history of any drug allergy.

After obtaining informed consent, patients were randomly assigned to one of the three treatment groups.

a. Ondansetron 150µg/kg
b. Granisetron 40µg/kg
c. Placebo (normal saline)

} after induction of anesthesia

by an anesthesiologist not involved in the assessment. A standardized anesthetic technique was followed

Premedication: Inj. Glycopyrolate 0.05mg/kg.

Inj. Pentazocine 0.5mg/kg.

i.m. 45 minutes before induction of anaesthesia.

After pre-oxygenation with 100% O_2 for 3 minutes, patients were induced with thiopentone 5mg/kg and suxamethonium 2mg/kg and intubated with appropriated sized endotracheal tubes. Patients were ventilated with IPPV with N_2O/O_2 2:1 and halothane (if needed, to maintain stable heamodynamics), paralysed with atracurium 0.5mg/kg initially, then 0.1mg/kg. At the end of surgery, patients were reversed with neostigmine 0.05mg/kg and glycopyrolate 0.1mg/kg and extubated. Intra-operatively ECG, HR, spo_2, NIBP were monitored. Intra-op and post-op fluids comprised of Ringer lactate based on 4-2-1 rule.

Post-operatively patients were assessed for nausea and vomiting in the early post-op period (upto 1 hour), upto oral intake and upto 24 hours. Presence of any side effects and

need for rescue anti-emetic (for more than 2 episodes of vomiting) were noted. Post-op pain was treated with acetaminophen suspension.

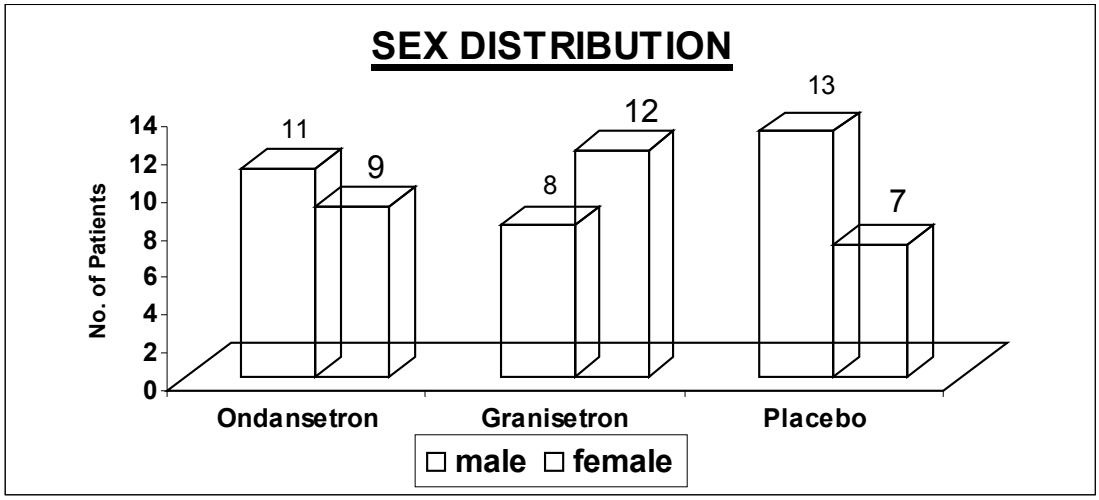
OBSERVATION AND RESULTS

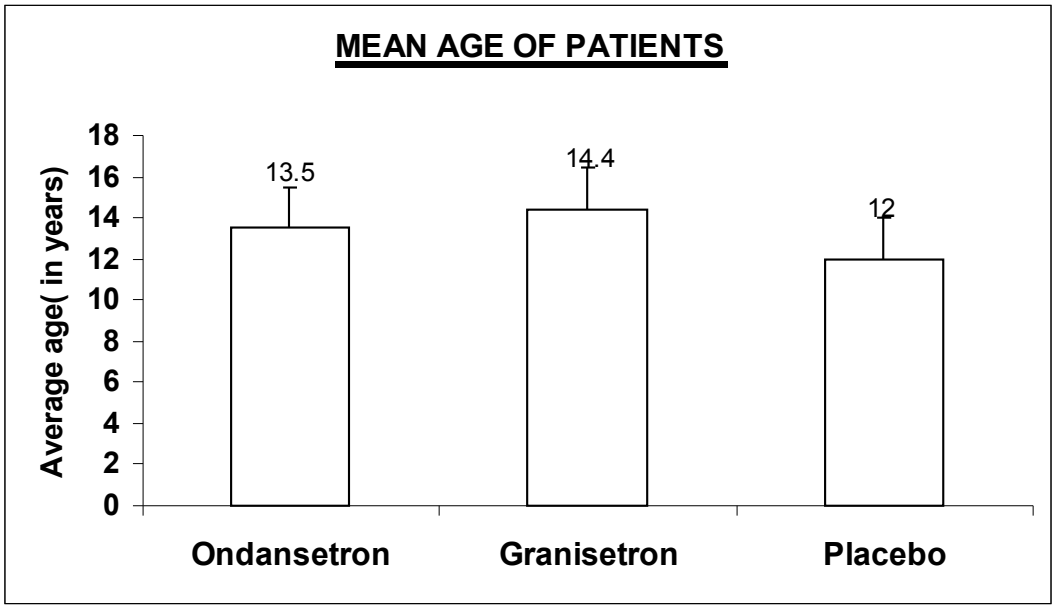
Demographic Profile

All parameters were comparable between the three groups as evaluated by ANOVA test.

Age

						F-test
Ondansetron	20	13.50	5.790	5	24	F=1.1 P=0.33
Granisetron	20	14.40	4.925	8	22	
Placebo	20	12.00	4.702	7	22	




MEAN AGE OF PATIENTS
Average age(in years)
18
16
14
12
10
8
6
4
2
0
13.5
14.4
12
Ondansetron
Granisetron
Placebo

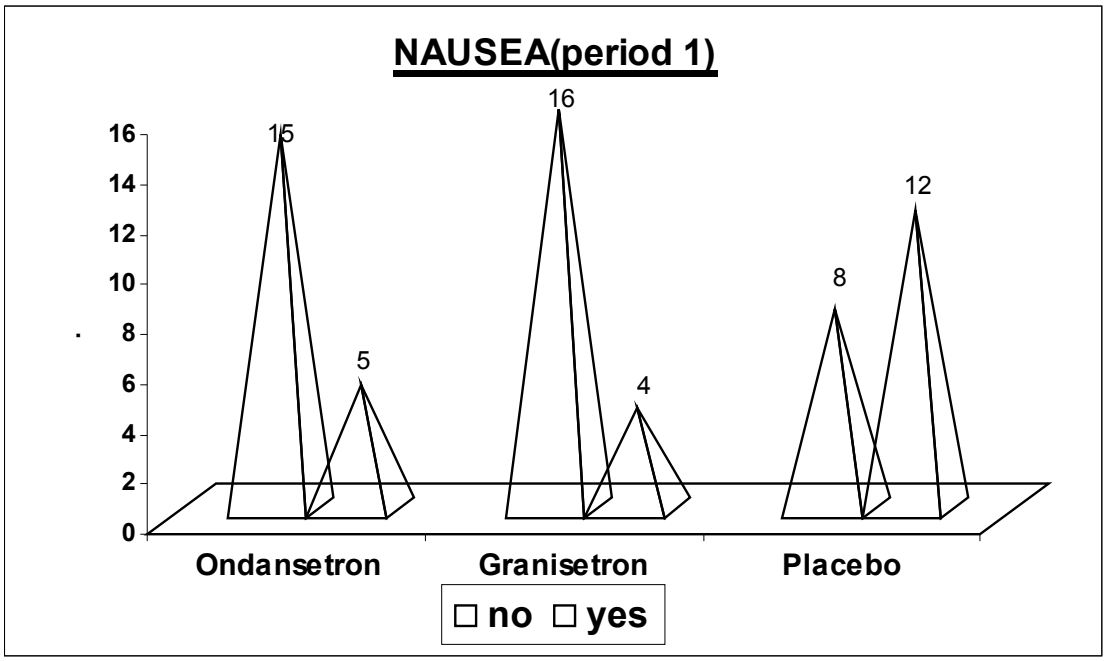
Observation

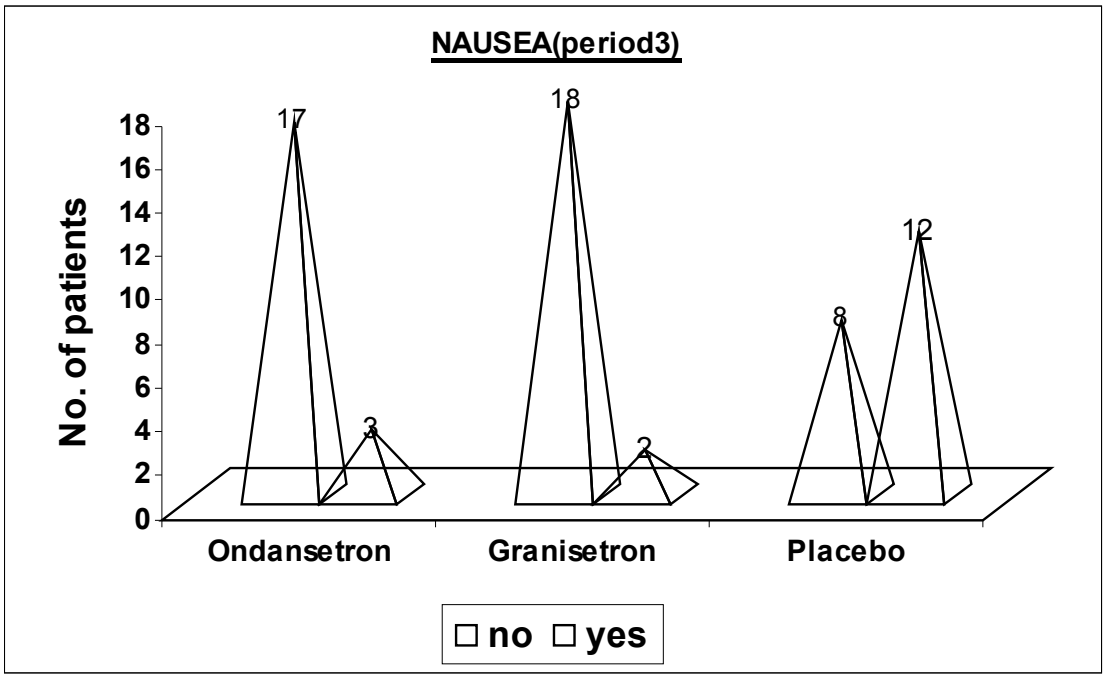
Nausea and vomiting were evaluated in three periods i.e. immediate post – op period (1 hr), upto oral intake and upto 24 hours using chi – square test. The presence of any side effects were also recorded.

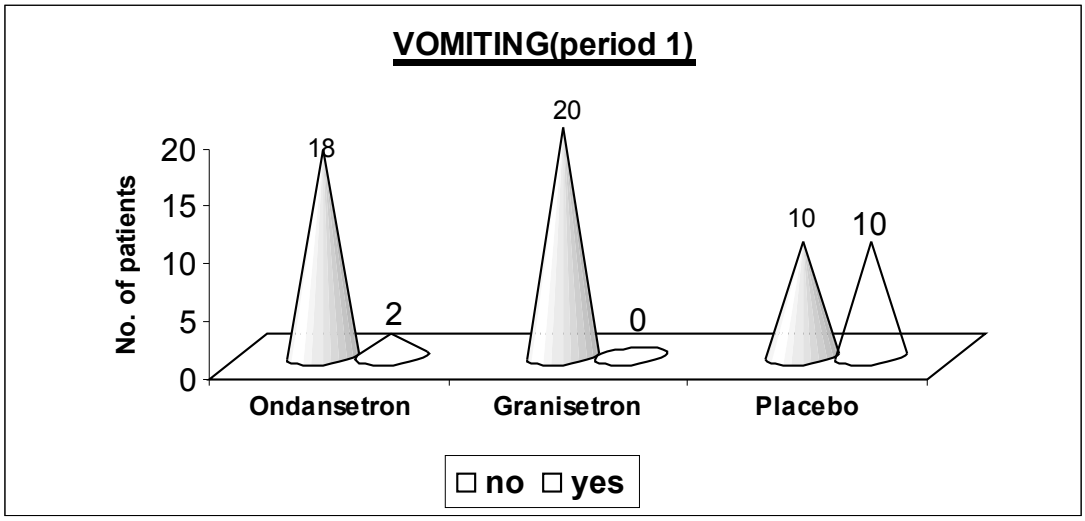
		group					
		Ondansetron		Granisetron		Placebo	
		n	%	n	%	n	%
sex	male	11	55.0%	8	40.0%	13	65.0%
	female	9	45.0%	12	60.0%	7	35.0%
surgery	Tonsil	14	70.0%	14	70.0%	14	70.0%
	Middle ear	6	30.0%	6	30.0%	6	30.0%
Nausea (period1)	no	15	75.0%	16	80.0%	8	40.0%
	yes	5	25.0%	4	20.0%	12	60.0%
Nausea (period2)	no	12	60.0%	14	70.0%	10	50.0%
	yes	8	40.0%	6	30.0%	10	50.0%
Nausea (period3)	no	17	85.0%	18	90.0%	8	40.0%
	yes	3	15.0%	2	10.0%	12	60.0%
vomiting (period1)	no	18	90.0%	20	100.0%	10	50.0%
	yes	2	10.0%	-	-	10	50.0%
vomiting (period2)	no	19	95.0%	18	90.0%	14	70.0%
	yes	1	5.0%	2	10.0%	6	30.0%
vomiting (period3)	no	19	95.0%	20	100.0%	13	65.0%
	yes	1	5.0%	-	-	7	35.0%
Rescue	no	20	100.0%	20	100.0%	13	65.0%
	yes	-	-	-	-	7	35.0%
headache	no	14	70.0%	15	75.0%	16	80.0%
	yes	6	30.0%	5	25.0%	4	20.0%
Abdominal discomfort	no	17	85.0%	19	95.0%	20	100.0%
	yes	3	15.0%	1	5.0%		
allergy	no	20	100.0%	20	100.0%	20	100.0%

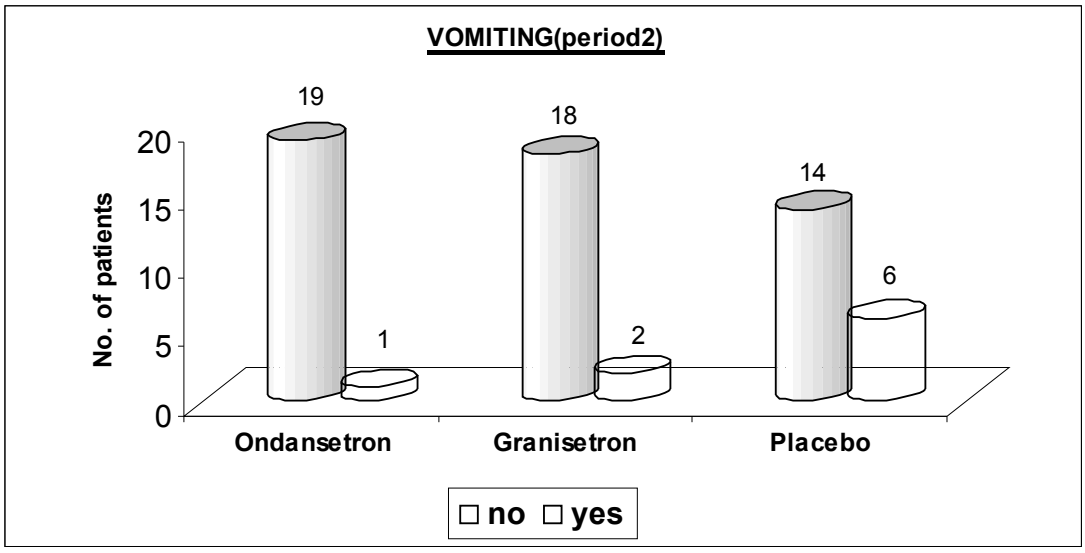
	χ2 test	P-value
nauseia (Period 1)	8.4	0.02
nauseia (Period 2)	1.7	0.43
nauseia (Period 3)	14.9	0.001
vomiting (Period 1)	17.5	0.001
vomiting (Period 2)	5.5	0.06
vomiting (Period 3)	12.4	0.002
rescue	15.8	0.001
headache	0.5	0.77

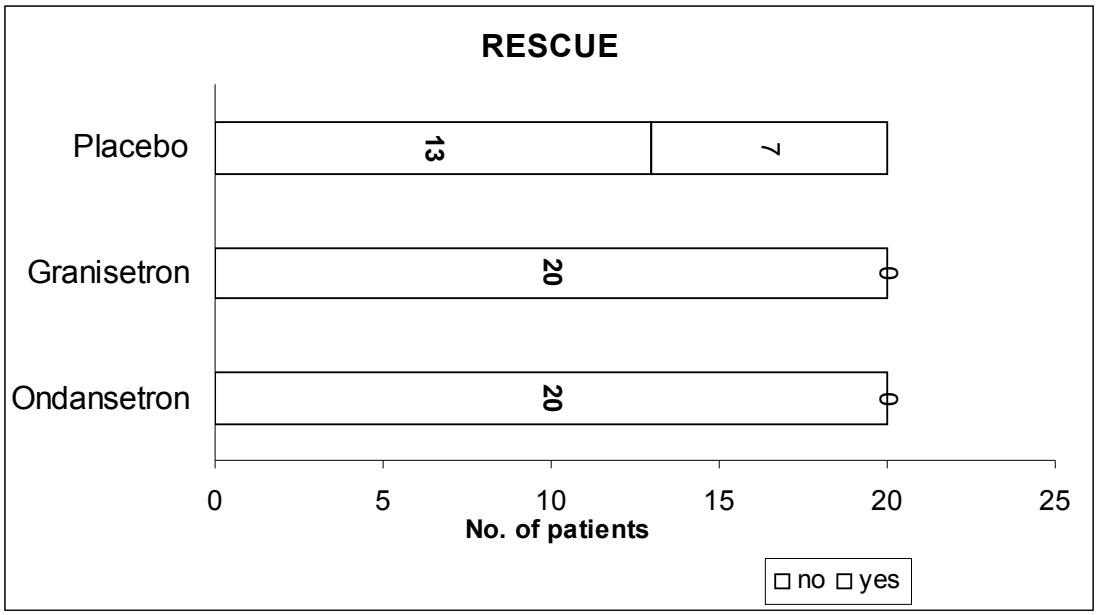
abdominal discomfort 3.75 0.15

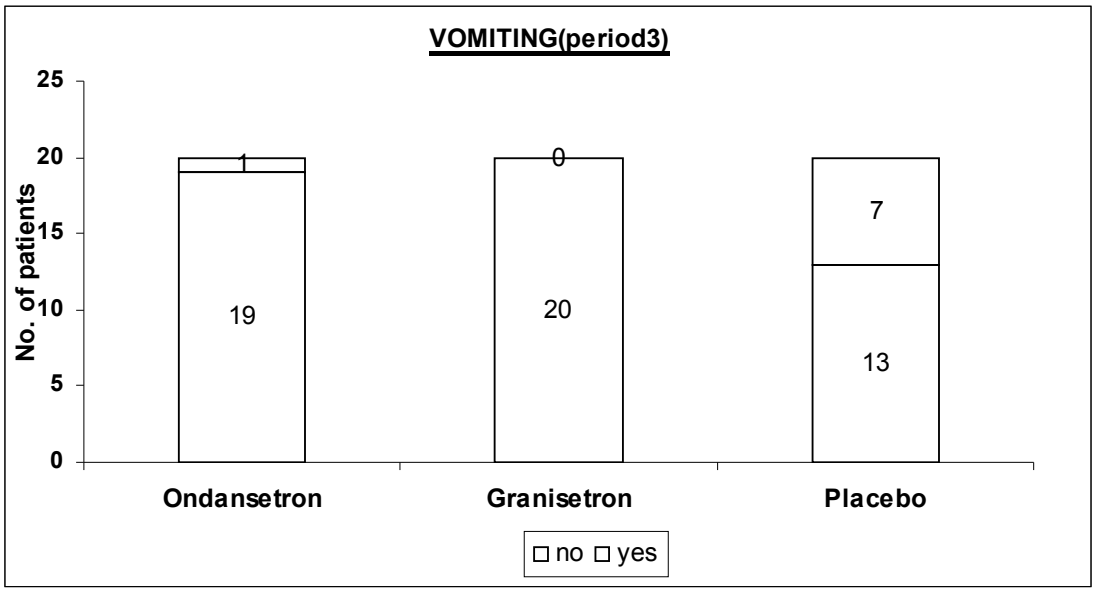



NAUSEA(period3)
No. of patients
18
16
14
12
10
8
6
4
2
0
17
3
18
2
8
12
Ondansetron
Granisetron
Placebo
no
yes


VOMITING(period 1)
No. of patients
20
15
10
5
0
18
2
20
0
10
10
Ondansetron
Granisetron
Placebo
no
yes


VOMITING(period2)
19
1
18
2
14
6
20
15
10
5
0
No. of patients
Ondansetron
Granisetron
Placebo
no
yes


RESCUE
Placebo
Granisetron
Ondansetron
13
7
20
0
20
0
0
5
10
15
20
25
No. of patients
no
yes



It is observed that the incidence of nausea and vomiting was significantly less between ondansetron, and granisetron group compared with placebo group in all the periods except for the incidence of nausea (Period 2). There was no statistically significant difference in the incidence of side effect.

	Nausea(period1)				Odds ratio
	no		yes		
	n	%	n	%	
Ondansetron	15	38.5%	5	23.8%	1.00
Granisetron	16	41.0%	4	19.0%	0.75
Placebo	8	20.5%	12	57.1%	4.5

$\chi 2$ test for trend = 5.2 P=0.02

	Nausea(period3)				Odds ratio
	no		yes		
	n	%	n	%	
Ondansetron	17	39.5%	3	17.6%	1.00
Granisetron	18	41.9%	2	11.8%	0.63
Placebo	8	18.6%	12	70.6%	3.78

$\chi 2$ test for trend = 3.97 P=0.05

	Vomiting (period1)				Odds ratio
	no		yes		
	n	%	n	%	
Ondansetron	18	37.5%	2	16.7%	1.00
Granisetron	20	41.7%	-	-	0.00
Placebo	10	20.8%	10	83.3%	9.00

$\chi 2$ test for trend = 9.8 P=0.001

	Vomiting (period2)				Odds ratio
	no		yes		
	n	%	n	%	
Ondansetron	19	37.3%	1	11.1%	1.00
Granisetron	18	35.3%	2	22.2%	2.11
Placebo	14	27.5%	6	66.7%	8.14

$\chi 2$ test for trend = 4.8 P=0.05

	Vomiting(period3)				Odds ratio
	no		yes		
	n	%	n	%	
Ondansetron	19	36.5%	1	12.5%	1.00
Granisetron	20	38.5%	-	-	0.00
Placebo	13	25.0%	7	87.5%	10.23

$\chi 2$ test for trend = 7.6 P=0.005

From the above values the odds ratio (Risk ratio) has been calculated. The risk of getting nausea and vomiting is highest in placebo group and least is granisetron group.

	rescue			
	no		yes	
	n	%	n	%
Ondansetron	20	37.7%	-	-
Granisetron	20	37.7%	-	-
Placebo	13	24.5%	7	100.0%

No patient in the ondansetron and granisetron group needed rescue antiemetics, where as nearly 65% in the placebo group required rescue drug.

DISCUSSION

Nausea and vomiting are both unpleasant and distressing to the patient, surgeon and anaesthesiologist. If severe, it can lead to separation of suture lines, wound dehiscence dehydration, electrolyte imbalance, exhaustion delayed discharge from hospital and increased cost to the patient.

Numerous drugs have been used in the past in the prevention of post – operative nausea and vomiting, but they also have been associated with undesirable side effects. For example, metaclopramide results in extrapyramidal symptoms, droperidol produces restlessness and dysphoric reactions, antihistaminics result in sedation. The 5 HT3 antagonists are very effective in preventing post-operative nausea and vomiting and do not produce any significant side effects. This study compares the efficacy of ondansetron and granisetron in the prevention of post – operative nausea and vomiting.

Fuji Y et al have done two studies in patients undergoing tonsillectomy and middle ear surgery comparing granisetron and placebo. The incidence of PONV in their studies were 17% and 60% in tonsillectomies and 17% and 63% in middle ear surgeries. In our study it was 30% and 85% respectively.

Morton NS et al compared ondansetron and placebo in patients undergoing tonsillectomy and demonstrated the superiority of ondansetron. The incidence of nausea was 36% and 49% and the incidence of vomiting was 40% and 53%. In our study it was 40% and 85% respectively.

Dua N et al compared granisetron and ondansetron for the prevention of nausea and vomiting in patients undergoing modified radical mastectomy and demonstrated that the incidence of PONV with ondensetron granisetron and placebo were 25%, 20% and 70% respectively. Our study findings

concurred with this study with a slightly higher incidence, the figures being 40%, 30% and 85% respectively.

No clinically significant side effects were noted with both these drugs during our study.

SUMMARY

This study was conducted in Madras Medical College to compare the efficacy of ondansetron and granisetron in the prevention of post – operative nausea and vomiting. Sixty patients in the age group of 5 – 25 years and ASA I or II status were chosen and randomly allotted to be in each of the following treatment group.

a) Inj ondansetron

b) Inj Granisetron

c) Placebo

After obtaining informed consent, the patients received the drugs after induction of anaesthesia. The anaesthetic technique was standardized. The patients were assessed for the incidence of nausea and vomiting upto 24 hours.

We found that both ondansetron and granisetron were effective in preventing nausea and vomiting with no clinically significant side effects.

CONCLUSION

We compared the efficacy of ondansetron and granisetron in patients undergoing tonsillectomy and middle ear surgery and found that both drugs were effective in preventing post – operative nausea and vomiting. There was a decrease in the requirements of rescue antiemetics, when these drugs were given. The side effects observed with these drugs were mild and clinically insignificant.

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PROFORMA

Name : No:

Age / Sex : Diagnosis :

Weight : Procedure :

ASA Status : Investigations :

Group : A) Inj. Ondansetron 150 µg / kg

B) Inj. Granisetron 40 µg/kg

C) Placebo

After induction of anaesthesia

	Immediate Post – Op Period (upto 1 hour)	Upto Oral intake	Upto 24 hours
Incidence of Nausea			
Incidence of vomiting			

Duration of anaesthesia :

Oral fluids started at :

Rescue antiemetic :

Side effects :

Head ache :

Abdominal discomfort :

Rash / Allergy :

MASTER CHART

S.NO	NAME	SEX / AGE	SURGERY	GROUP	NAUSEA Period			VOMITING Period			RESCUE ANTI -EMETICS	HEAD ACHE	ABDOMINAL. DISCOMFORT	RASH/ ALLERGY
					1	2	3	1	2	3				
1.	VIGNESWAR	M/10	TONSIL	ONDANSETRON	-	-	-	-	-	-	-	-	-	-
2.	SUJATHA	F/9	TONSIL	ONDANSETRON	-	-	-	-	-	-	-	+	-	-
3.	AMUTHA	F/9	TONSIL	ONDANSETRON	+	+	+	+	-	-	-	-	+	-
4.	DIVYA	F/5	TONSIL	ONDANSETRON	-	-	-	-	-	-	-	-	-	-
5.	PRAKASH	M/7	TONSIL	ONDANSETRON	-	-	-	-	-	-	-	+	-	-
6.	MALATHY	F/9	TONSIL	ONDANSETRON	+	+	-	-	-	-	-	+	-	-
7.	JOTHI	F/12	TONSIL	ONDANSETRON	-	-	-	-	-	-	-	-	-	-
8.	LINGESWARAN	M/9	TONSIL	ONDANSETRON	-	-	-	-	-	-	-	-	-	-
9.	GOPI	M/11	TONSIL	ONDANSETRON	+	+	+	+	-	-	-	-	-	-
10.	NASREEN	F/12	TONSIL	ONDANSETRON	-	-	-	-	-	-	-	-	-	-
11.	SURESH	M/14	TONSIL	ONDANSETRON	-	-	-	-	-	-	-	+	+	-
12.	FATHIMA	F/20	TONSIL	ONDANSETRON	+	+	-	-	-	-	-	-	-	-
13.	SEKAR	M/13	TONSIL	ONDANSETRON	-	-	-	-	-	-	-	-	-	-
14.	THARSEIVEL	M/12	TONSIL	ONDANSETRON	+	+	-	-	+	+	-	-	-	-
15.	NAGARAJ	M/24	ME	ONDANSETRON	-	-	-	-	-	-	-	-	+	-
16.	CHANDRAN	M/22	ME	ONDANSETRON	-	+	+	-	-	-	-	+	-	-
17.	PARVATHY	F/22	ME	ONDANSETRON	-	-	-	-	-	-	-	-	-	-
18.	DEEPAK	M/11	ME	ONDANSETRON	-	-	-	-	-	-	-	-	-	-
19.	ANANDHI	F/15	ME	ONDANSETRON	-	+	+	-	-	-	-	-	-	-
20.	RAMESH	M/24	ME	ONDANSETRON	-	-	-	-	-	-	-	+	-	-

MASTER CHART

S.NO	NAME	SEX / AGE	SURGERY	GROUP	NAUSEA Period			VOMITING Period			RESCUE ANTI-EMETICS	HEAD ACHE	ABDOMINAL. DISCOMFORT	RASH/ ALLERGY
					1	2	3	1	2	3				
21.	GEETHA	F/19	TONSIL	GRANISETRON	-	-	-	-	-	-	-	-	-	-
22.	MANIKANDAN	M/12	TONSIL	GRANISETRON	-	-	-	-	-	-	-	-	-	-
23.	KOTHAI	M/21	TONSIL	GRANISETRON	+	+	+	-	-	-	-	+	-	-
24.	RAJKUMAR	M/12	TONSIL	GRANISETRON	-	-	-	-	-	-	-	-	-	-
25.	DAS	M/22	TONSIL	GRANISETRON	-	-	-	-	-	-	-	-	-	-
26.	DINESH	M/12	TONSIL	GRANISETRON	+	+	-	-	-	-	-	+	+	-
27.	SELVI	F/12	TONSIL	GRANISETRON	-	-	-	-	-	-	-	+	-	-
28.	ANJALI	F/11	TONSIL	GRANISETRON	-	-	-	-	-	-	-	-	-	-
29.	RUBINI	F/10	TONSIL	GRANISETRON	-	-	-	-	-	-	-	-	-	-
30.	MANJULA	F/9	TONSIL	GRANISETRON	+	+	+	-	+	-	-	-	-	-
31.	VANITHA	F/9	TONSIL	GRANISETRON	-	-	-	-	-	-	-	-	-	-
32.	MOHARAPRIYA	F/8	TONSIL	GRANISETRON	-	-	-	-	-	-	-	+	-	-
33.	GRACY	F/12	TONSIL	GRANISETRON	-	-	-	-	-	-	-	-	-	-
34.	SUDHA	F/9	TONSIL	GRANISETRON	+	+	-	-	-	-	-	-	-	-
35.	SHARTNA	F/14	ME	GRANISETRON	-	-	-	-	-	-	-	-	-	-
36.	SAGAYAM	F/22	ME	GRANISETRON	-	-	-	-	-	-	-	-	-	-
37.	BALAMMESAR	M/22	ME	GRANISETRON	-	+	-	-	+	-	-	-	-	-
38.	SARAVANAN	M/15	ME	GRANISETRON	-	-	-	-	-	-	-	-	-	-
39.	SURESH	M/19	ME	GRANISETRON	-	-	-	-	-	-	-	+	-	-
40.	RAJI	M/18	ME	GRANISETRON	-	+	-	-	-	-	-	-	-	-

MASTER CHART

S.NO	NAME	SEX / AGE	SURGERY	GROUP	NAUSEA Period			VOMITING Period			RESCUE ANTI -EMETICS	HEAD ACHE	ABDOMINAL DISCOMFORT	RASH/ ALLERGY
					1	2	3	1	2	3				
41.	NARESH	M/9	TONSIL	PLACEBO	-	-	+	-	-	+	-	-	-	-
42.	VIGNESH	M/10	TONSIL	PLACEBO	+	+	+	+	-	+	+	-	-	-
43.	BHUVANESH	M/10	TONSIL	PLACEBO	-	-	-	-	-	-	-	-	-	-
44.	JOSEPH	M/7	TONSIL	PLACEBO	-	-	-	-	-	-	-	+	-	-
45.	ARUNKUMAR	M/8	TONSIL	PLACEBO	+	+	-	+	+	-	-	+	-	-
46.	SAKTHI	F/8	TONSIL	PLACEBO	-	-	+	-	-	+	-	-	-	-
47.	SAROJA	F/10	TONSIL	PLACEBO	+	+	+	+	-	+	+	-	-	-
48.	PREMKUMAR	M/8	TONSIL	PLACEBO	+	+	+	+	-	+	+	-	-	-
49.	SASIKALA	F/10	TONSIL	PLACEBO	-	+	-	-	+	-	-	-	-	-
50.	POOVARNAM	M/10	TONSIL	PLACEBO	+	-	+	-	-	-	-	-	-	-
51.	VIJAYBABU	M/10	TONSIL	PLACEBO	+	+	+	+	+	+	+	+	-	-
52.	RISWAS	F/12	TONSIL	PLACEBO	-	-	-	-	-	-	-	-	-	-
53.	DINESH	M/12	TONSIL	PLACEBO	+	-	-	-	-	-	-	-	-	-
54.	REVATHY	F/11	TONSIL	PLACEBO	+	+	+	+	+	-	+	-	-	-
55.	KANNAN	M/10	ME	PLACEBO	-	-	+	-	-	-	-	-	-	-
56.	SENANRAJ	M/10	ME	PLACEBO	+	+	+	+	+	-	+	-	-	-
57.	GOWRI	F/18	ME	PLACEBO	-	+	-	-	+	-	-	+	-	-
58.	PERUMAL	M/22	ME	PLACEBO	+	-	+	+	-	-	-	-	-	-
59.	MOHAN	M/22	ME	PLACEBO	+	-	+	+	+	+	+	-	-	-
60.	CHITHRA	F/21	ME	PLACEBO	+	-	-	+	-	-	-	-	-	-